10/670,065
LY Cook 4/28/07
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(FILE 'HOME' ENTERED AT 19:47:25 ON 28 APR 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 19:47:42 ON 28 APR 2007

L1	1995	S MDM AND MACRO?
L2	9	S L1 AND VIMENTIN?
L3	6	DUPLICATE REMOVE L2 (3 DUPLICATES REMOVED)
L4	27	S L1 AND COLI?
L5	14	DUPLICATE REMOVE L4 (13 DUPLICATES REMOVED)
L6	7	S L5 AND PD<2003
L7	17811	S (OKADAIC ACID)
L8	0	S L6 AND L7
L9	343	S GO6983?
L10	0	S L9 AND L6
L11	0	S L9 AND L1
L12	3	S L9 AND VIMENTIN?
L13	1	DUPLICATE REMOVE L12 (2 DUPLICATES REMOVED)
L14	12	S L9 AND BACTE?
L15	642	DUPLICATE REMOVE L1 (1353 DUPLICATES REMOVED)
L16	7	DUPLICATE REMOVE L14 (5 DUPLICATES REMOVED)
L17	1	S L16 AND PD<2003
L18	142	S L7 AND VIMENTIN?
L19	6097	S (SB 203580)
L20	10	S L19 AND VIMENTIN?
L21	10	DUPLICATE REMOVE L20 (0 DUPLICATES REMOVED)
L22	5	S L21 AND PD<2003
L23	12	S L9 AND BACTER?
L24	7	DUPLICATE REMOVE L23 (5 DUPLICATES REMOVED)
L25	1	S L24 AND PD<2003

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L7         17811 S (OKADAIC ACID)
L8             0 S L6 AND L7
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L22            5 S L21 AND PD<2003
L23           12 S L9 AND BACTER?
L24            7 DUPLICATE REMOVE L23 (5 DUPLICATES REMOVED)
L25            1 S L24 AND PD<2003

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ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2001:437639 BIOSIS
DN PREV200100437639
TI Dual actions of Pasteurella multocida toxin (Galphaq agonist) to promote cardiomyocyte hypertrophy and enhance susceptibility to apoptosis.
AU Sabri, Abdelkarim [Reprint author]; Wilson, Brenda A.; Steinberg, Susan F.
CS Columbia University, New York, NY, USA
SO Journal of Molecular and Cellular Cardiology, (June, 2001) Vol. 33, No. 6, pp. A104. print.
Meeting Info.: XVII ISHR World Congress of the International Society for Heart Research. Winnipeg, Canada. July 06-11, 2001. International Society for Heart Research.
CODEN: JMCDAY. ISSN: 0022-2828.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 12 Sep 2001
Last Updated on STN: 22 Feb 2002
CC General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - General and comparative studies: coenzymes 10802
Cardiovascular system - Physiology and biochemistry 14504
Endocrine - General 17002
Toxicology - General and methods 22501
Physiology and biochemistry of bacteria 31000
IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics); Toxicology; Cardiovascular System (Transport and Circulation)
IT Parts, Structures, & Systems of Organisms
cardiac fibroblast: circulatory system; cardiac sarcomere: circulatory system, muscular system; heart: circulatory system
IT Diseases
cardiomyocyte hypertrophy: heart disease, pathogenesis
IT Chemicals & Biochemicals
AKT: phosphorylation; ANF [atrial natriuretic factor]: expression; ERK [extracellular signal-regulated kinase]: activation; Go6983: enzyme inhibitor; Gq protein alpha subunit: expression, regulation; JNK [c-Jun N-terminal kinase]: activation; Pasteurella multocida toxin: Gq protein alpha agonist, toxin; epidermal growth factor receptor [EGF receptor]: activation; p38-MAPK [p38-mitogen-activated protein kinase]: activation; phospholipase C: activation; protein kinase C: isoforms, novel, regulation
IT Miscellaneous Descriptors
apoptosis susceptibility modulation; cardiomyocyte apoptosis; Meeting Abstract
ORGN Classifier
Pasteurellaceae 06703
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria; Microorganisms
Organism Name
Pasteurella multocida: toxin source
Taxa Notes
Bacteria, Eubacteria, Microorganisms
RN 133053-19-7 (Go6983)
37333-47-4 (Pasteurella multocida toxin)
9001-86-9Q (phospholipase C)
63551-76-8Q (phospholipase C)
141436-78-4 (protein kinase C)
85637-73-6 (ATRIAL NATRIURETIC FACTOR)
142243-02-5 (EXTRACELLULAR SIGNAL-REGULATED KINASE)
155215-87-5 (C-JUN N-TERMINAL KINASE)
165245-96-5 (P38-MITOGEN-ACTIVATED PROTEIN KINASE)

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THE STUDY ON THE RELATIONSHIP BETWEEN HUMAN CYTOMEGALOVIRUS INFECTION AND ATHEROSCLEROSIS DEVELOPMENT

Chen Ruizhen, Yang Yingzhen, Fu Weiguo, Wang Yuqi, Ge Junbo.(Key Laboratory of Viral Heart Diseases of Ministry of Public Health; Shanghai Institute of Cardiovascular Diseases; Zhongshan Hospital, Fudan University, Shanghai 200032 China)

To investigate the viral etiological factor of Atherosclerosis (AS). Artery vascular tissue specimens derived from 75 patients with AS were studied for detection of human cytomegalovirus (HCMV) immediately early (IE) and later (L) gene fragments by polymerase chain reaction (PCR) and in situ hybridization; sera collected from the same patients were examined for HCMV specific IgG and IgM by enzyme-linked immunosorbent assay (ELISA). Twenty two normal arterial tissues and sera were used as controls. The positive rate of HCMV L and IE gene fragments were significantly higher in As patients than those in controls. HCMV DNA were mainly observed in nucleus of endothelial cells, muscularis under intima and smooth muscle of media of As, rarely found in controls. There are some relationship between HCMV and AS; Artery itself maybe the site of HCMV latency. Meanwhile, Higher levels of HCMV-sIgG and sIgM were found to be associated virus, indicating that a periodically actived latent infection or a continuously active infection is presented in AS patients.

DUAL ACTIONS OF *PASTEURELLA MULTOCIDA* TOXIN (Gαq AGONIST) TO PROMOTE CARDIOMYOCYTE HYPERTROPHY AND ENHANCE SUSCEPTIBILITY TO APOPTOSIS

Abdelkarim Sabri, Brenda A. Wilson & Susan F. Steinberg Columbia University, NY & Univ IL at Urbana-Champaign

Previous attempts to delineate the consequences of Gq protein α subunit activation in cardiomyocytes relied largely on molecular strategies in cultures or transgenic mice. Modest levels of WT Gαq overexpression induce stable cardiac hypertrophy, whereas intense Gαq stimulation (with high levels of WT Gαq or the constitutively activated mutant) induces cardiomyocyte apoptosis. The precise mechanism(s) whereby traditional targets of Gαq that induce hypertrophy also trigger cardiomyocyte apoptosis is not obvious and is explored with recombinant *Pasteurella multocida* toxin (rPMT, a Gαq agonist). Chronic stimulation with rPMT leads to cardiomyocyte enlargement, sarcomeric organization, and increased ANF expression in association with activation of phospholipase C, novel protein kinase C (PKC) isoforms, ERK, and (to a lesser extent) JNK/p38-MAPK. rPMT stimulates the ERK cascade via epidermal growth factor (EGF) receptor transactivation in cardiac fibroblasts, but not in cardiomyocytes. Surprisingly, rPMT (or PKC activation by phorbol ester) *decreases* basal AKT phosphorylation and *prevents* AKT phosphorylation by EGF. The rPMT-dependent decrease in AKT phosphorylation is abrogated by Go6983 (PKC inhibitor) and is functionally significant; cardiomyocyte apoptosis is augmented in rPMT-treated cultures. These results link nPKC isoform activation by Gαq to reduced AKT phosphorylation, impaired AKT stimulation by survival pathways, and enhanced apoptosis susceptibility. AKT inhibition by PKC is predicted to contribute to the transition from hypertrophy to heart failure and represent a target for therapy.

INVOLVEMENT OF 1,2-DIACYLGLYCEROL IN CARDIAC HYPERTROPHY OF THE JUVENILE VISCERAL STEATOSIS MICE WITH SYSTEMIC CARNITINE DEFICIENCY

Yoshihiro Saburi, Hiroki Kamiya, Kazunori Hayashi, Hideo Matsui, Kenji Okumura

Background: The juvenile visceral steatosis (JVS) mouse, an animal model of primary systemic carnitine deficiency, is known to develop cardiac hypertrophy. This study was performed as to whether cardiac hypertrophy in JVS mice is associated with the quantitative and qualitative changes of 1,2-diacylglycerol (1,2-DAG), an activator of protein kinase C.
Methods: Controls and JVS mice were evaluated at 4 weeks (developing hypertrophy stage) and 8 weeks (established hypertrophy stage) of age. Myocardial 1,2-DAG level was measured by FID-TLC method and its fatty acid composition was analysed by gas chromatography.
Results: The heart weight/body weight ratio was 2.5-fold higher and left ventricle wall thickness was 1.4-fold higher in JVS mice at 8 weeks compared with those of controls ($p<0.01$, respectively). Myocardial 1,2-DAG level at 4 weeks was already elevated in JVS mice (1023 vs. 1366 ng/dry wt, $p=0.07$). At 8 weeks, the 1,2-DAG level was remarkably increased in JVS mice compared with that in controls (819 vs. 2063 ng/dry wt, $p<0.01$). Fatty acids of 18:1 and 18:2 were significantly elevated in hearts of JVS mice both at 4 and 8 weeks compared with those of controls ($P<0.01$, respectively).
Conclusion: Increase in distinct 1,2-DAG species containing 18:1 and 18:2 fatty acids might be involved in the pathogenesis of cardiac hypertrophy in JVS with systemic carnitine deficiency .

ENDOTHELIAL CELL DYSFUNCTION-KEY FACTOR IN ATHEROGENESIS AND ITS REVERSAL (LABORATORY & CLINICAL STUDY)

G S Sainani, Manisha Sawhney
Jaslok Hosp. & Research Centre, Mumbai (India)

In macrophage cell cultures and DNA studies, we recorded morphological changes and DNA damage caused by hydrogen peroxide exposure. In another experiment native LDL was converted to oxidised LDL by exposure to cigarette smoke extract. In another experiment, interaction of altered macrophages and OX-LDL resulted in foam cells. However pre-treatment with anti-oxidants-superoxide dismutase (SOD), nitric oxide (NO), glutathione peroxidase (Gpx,) vitamins E, C, β-carotene in all above, experiments reversed partially/completely oxidative damage of macrophages, DNA, LDL and prevented foam cell formation. Clinical study included 100 documented cases of coronary artery disease (CAD) (50-angiographically proved, 50 acute myocardial infarction patients and 100 healthy controls (angiographically negative). We estimated oxidative stress (thiobarbituric acid reactive substances and conjugated diene levels) and antioxidant levels (SOD, NO, Gpx, vitamins E,C,β-carotene, selenium) in all. Anti-oxidant levels were significantly low in CAD group. Low NO levels in CAD reflect endothelial cell dysfunction (ECD). Coronary artery lesions improved with treatment.
We conclude that ECD is the key factor in CAD and with timely treatment, ECD and coronary artery blocks can be reversed to variable extent.

10/670,065
L/Cook 4/28/07
Search Updated

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(FILE 'HOME' ENTERED AT 20:44:32 ON 28 APR 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 20:45:00 ON 28 APR 2007

L1	44	S	(VIMENTIN KINASE)
L2	42232	S	VIMENTIN?
L3	4643	S	L2 AND MONOCLONAL?
L4	832	S	L3 AND POLYCLONAL?
L5	4	S	L4 AND (OKADAIC ACID)
L6	1		DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)
L7	0	S	L2 AND ANTSENSE?
L8	237	S	L2 AND ANTISENSE?
L9	38	S	L8 AND VECTOR?
L10	22	S	L9 AND PD<2003
L11	25687	S	SIRNA
L12	81	S	L11 AND L2
L13	7	S	L12 AND L8
L14	0	S	L13 AND PD<2003
L15	8	S	L10 AND RNA?
L16	12	S	L9 AND RNA?
L17	1832	S	(THIOL PROTEINASE)
L18	43	S	(NEUTRAL THIOL PROTEINASE)
L19	15	S	L18 AND L2
L20	5		DUPLICATE REMOVE L19 (10 DUPLICATES REMOVED)
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L21            5 S L20 AND PD<2003
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AN 1988:480120 BIOSIS

DN PREV198886111430; BA86:111430

TI DIFFERENTIAL SENSITIVITY OF VIMENTIN AND NUCLEAR LAMINS FROM EHRLICH ASCITES TUMOR CELLS TOWARD CALCIUM-ACTIVATED NEUTRAL THIOL PROTEINASE.

AU TRAUB P [Reprint author]; SCHERBARTH A; WILLINGALE-THEUNE J; PAULIN-LEVASSEUR M; SHOEMAN R

CS MAX-PLANCK-INST ZELLBIOL, ROSENHOF, D-6802 LADENBURG, W GER

SO European Journal of Cell Biology, (1988) Vol. 46, No. 3, pp. 478-490.
CODEN: EJCBDN. ISSN: 0171-9335.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 1 Nov 1988
Last Updated on STN: 1 Nov 1988

AB A comparative study of the susceptibility of vimentin and nuclear lamins from cultured Ehrlich ascites tumor (EAT) cells to degradation of Ca2+-activated neutral thiol proteinase (calpain) has been undertaken. While pure vimentin was degraded very quickly at physiological ionic strength by putrified calpain, isolated lamin B was digested comparatively slowly and purified lamins A/C were fairly resistant to proteolytic degradation. Similar digestion patterns were obtained from vimentin and lamin B with intermediary breakdown products close in size to the corresponding α-helical rod domains. The exclude the possibility that the low susceptibility of isolated lamins to Ca2+-dependent proteolytic degradation was due to irreversible denaturation during their isolation and purification, Triton cytoskeletons were prepared and their nuclear lamina as well as vimentin filaments were exposed to relatively large quantities of purified calpain. Under these conditions, not only vimentin filaments but also lamins A and B were digested while lamin C remained intact to a high degree. The major breakdown products of vimentin and lamins were identified as polypeptides which were 35 to 45 amino acids longer than the corresponding α-helical rod domains. Most of the vimentin-derived material and all high molecular weight polypeptides originating from lamins remained associated with the Triton cytoskeletons as demonstrated by sodium dodecyl sulfate polyacrylamide gel electophoresis in conjunction with immunoblotting. Indirect immunofluorescence and electron microscope analysis of the calpain-digested Triton cytoskeletons revealed that they still contained a laminalike structure around the nuclear chromatin and numerous structurally altered intermediate filaments in the cytoplasmic remnant, although all vimentin had been degraded with the formation of 40/41 kDa polypeptides as major digestion products. In untreated Triton cytoskeletons, the vimentin filaments seemed to be in direct physical contact with the nuclear lamina, whereas in digested Triton cytoskeletons there was a distinct gap between structurally altered filaments and the nuclear surface. This shows that vimentin filaments and the nuclear lamina are differently susceptible to degradation by calpain under certain ionic conditions and suggests that both filamentous structures are intimately associated with each other. Moreover, the large differences in isoelectric point between lamins A/C and lamin B as well as the high degee of microheterogeneity of lamins isolated from proliferating EAT cells might result primarily from structural differences in the α-helical rod domains of these karyoskeletal proteins.

CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012

IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Enzymology

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AB A comparative study of the susceptibility of vimentin and nuclear lamins from cultured Ehrlich ascites tumor (EAT) cells to degradation of Ca2+-activated neutral thiol proteinase (calpain) has been undertaken. While pure vimentin was degraded very quickly at physiological ionic strength by putrified calpain, isolated lamin B was digested comparatively slowly and purified lamins A/C were fairly resistant to proteolytic degradation. Similar digestion patterns were obtained from vimentin and lamin B with intermediary breakdown products close in size to the corresponding α-helical rod domains. The exclude the possibility that the low susceptibility of isolated lamins to Ca2+-dependent proteolytic degradation was due to irreversible denaturation during their isolation and purification, Triton cytoskeletons were prepared and their nuclear lamina as well as vimentin filaments were exposed to relatively large quantities of purified calpain. Under these conditions, not only vimentin filaments but also lamins A and B were digested while lamin C remained intact to a high degree. The major breakdown products of vimentin and lamins were identified as polypeptides which were 35 to 45 amino acids longer than the corresponding α-helical rod domains. Most of the vimentin-derived material and all high molecular weight polypeptides originating from lamins remained associated with the Triton cytoskeletons as demonstrated by sodium dodecyl sulfate polyacrylamide gel electophoresis in conjunction with immunoblotting. Indirect immunofluorescence and electron microscope analysis of the calpain-digested Triton cytoskeletons revealed that they still contained a laminalike structure around the nuclear chromatin and numerous structurally altered intermediate filaments in the cytoplasmic remnant, although all vimentin had been degraded with the formation of 40/41 kDa polypeptides as major digestion products. In untreated Triton cytoskeletons, the vimentin filaments seemed to be in direct physical contact with the nuclear lamina, whereas in digested Triton cytoskeletons there was a distinct gap between structurally altered filaments and the nuclear surface. This shows that vimentin filaments and the nuclear lamina are differently susceptible to degradation by calpain under certain ionic conditions and suggests that both filamentous structures are intimately associated with each other. Moreover, the large differences in isoelectric point between lamins A/C and lamin B as well as the high degee of microheterogeneity of lamins isolated from proliferating EAT cells might result primarily from structural differences in the α-helical rod domains of these karyoskeletal proteins.

CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012

IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Enzymology

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AN 1988:480120 BIOSIS

DN PREV198886111430; BA86:111430

TI DIFFERENTIAL SENSITIVITY OF VIMENTIN AND NUCLEAR LAMINS FROM EHRLICH ASCITES TUMOR CELLS TOWARD CALCIUM-ACTIVATED NEUTRAL THIOL PROTEINASE.

AU TRAUB P [Reprint author]; SCHERBARTH A; WILLINGALE-THEUNE J; PAULIN-LEVASSEUR M; SHOEMAN R

CS MAX-PLANCK-INST ZELLBIOL, ROSENHOF, D-6802 LADENBURG, W GER

SO European Journal of Cell Biology, (1988) Vol. 46, No. 3, pp. 478-490.
CODEN: EJCBDN. ISSN: 0171-9335.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 1 Nov 1988
Last Updated on STN: 1 Nov 1988

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Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012

IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Enzymology

(Biochemistry and Molecular Biophysics)
IT Miscellaneous Descriptors
MOUSE CALPAIN INTERMEDIATE FILAMENT CYTOSKELETON CELL CYCLE
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
RN 7440-70-2 (CALCIUM)
37353-41-6 (THIOL PROTEINASE)
78990-62-2 (CALPAIN)

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AN 1987:104624 BIOSIS
DN PREV198783053602; BA83:53602
TI PROTEIN-CHEMICAL IDENTIFICATION OF THE MAJOR CLEAVAGE SITE OF THE CALCIUM PROTEINASE ON MURINE VIMENTIN THE MESENCHYMAL INTERMEDIATE FILAMENT PROTEIN.
AU FISCHER S [Reprint author]; VANDEKERCKHOVE J; AMPE C; TRAUB P; WEBER K
CS MAX-PLANCK-INST BIOPHYS CHEM, POSTFACH 2841, D-3400 GOETTINGEN, W GER
SO Biological Chemistry Hoppe-Seyler, (1986) Vol. 367, No. 11, pp. 1147-1152.
CODEN: BCHSEI. ISSN: 0177-3593.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 26 Feb 1987
Last Updated on STN: 26 Feb 1987
AB Neutral thiol proteinases (calpains), activated by calcium are involved in the intracellular turnover of intermediate filaments but the precise position of the cleavage points has remained unknown. Here we identify by direct sequence analysis the major cleavage sites found when murine vimentin is digested by limited proteolysis in vitro with calpain purified from porcine kidney. Contrary to some previous suggestions, no absolute sequence specificity could be detected although 10 specific sites have been identified. This result is in line with cDNA derived amino-acid sequence of a calpain, which pointed to a similarity of the catalytic site with the active sites in papain, cathepsin and actinidin. However, all major cleavage sites are located within regions of the vimentin molecule, which in current models of intermediate filament structure are thought to be non-helical: the amino-terminal headpiece, the carboxyl-terminal tail-piece and the spacer separating the two major coiled-coil domains. The sequence information about the cleavage sites was extended to provide the amino-terminal 119 residues of murine vimentin.
CC Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Minerals 10069
Biophysics - Biocybernetics 10515
Enzymes - General and comparative studies: coenzymes 10802
Enzymes - Chemical and physical 10806
IT Major Concepts
Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and Molecular Biophysics)
IT Miscellaneous Descriptors
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Super Taxa
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Biophysics - Biocybernetics 10515
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Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and Molecular Biophysics)
IT Miscellaneous Descriptors
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ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1986:357972 BIOSIS
DN PREV198682062446; BA82:62446
TI EFFICIENT DEGRADATION IN-VITRO OF ALL INTERMEDIATE FILAMENT SUBUNIT PROTEINS BY THE CALCIUM-ACTIVATED NEUTRAL THIOL PROTEINASE FROM EHRLICH ASCITES TUMOR CELLS AND PORCINE KIDNEY.
AU VORGIAS C E [Reprint author]; TRAUB P
CS MAX-PLANCK-INST ZELLBIOL, ROSENHOF, D-6802 LADENBURG/HEIDELBERG, FRG
SO Bioscience Reports, (1986) Vol. 6, No. 1, pp. 57-64.
CODEN: BRPTDT. ISSN: 0144-8463.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 6 Sep 1986
Last Updated on STN: 6 Sep 1986
AB Vimentin, desmin, glial fibrillary acidic protein, neurofilament triplet proteins, and a mixture of cytokeratins were digested with Ca2+-activated neutral thiol proteinase isolated from Ehrlich ascites tumor (EAT) cells and porcine kidney. All intermediate filament proteins were degraded by the proteinase, although with different rates and Ca2+ optima. These results are in part at variance with our previous statement that the Ca2+-activated proteinase from EAT cells is specific for vimentin and desmin.
CC Cytology - Animal 02506
Biochemistry studies - Minerals 10069
Enzymes - Physiological studies 10808
Metabolism - Proteins, peptides and amino acids 13012
Urinary system - Physiology and biochemistry 15504
Neoplasms - Neoplastic cell lines 24005
In vitro cellular and subcellular studies 32600
IT Major Concepts
Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Metabolism
IT Miscellaneous Descriptors
VIMENTIN DESMIN GLIAL FIBRILLARY ACIDIC PROTEIN NEUROFILAMENT TRIPLET PROTEINS CYTOKERATINS
ORGN Classifier
Suidae 85740
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In vitro cellular and subcellular studies 32600
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In vitro cellular and subcellular studies 32600
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RN 7440-70-2 (CALCIUM)
37353-41-6 (THIOL PROTEINASE)

ANSWER 5 OF 5 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1984:81710 CAPLUS
DN 100:81710
ED Entered STN: 12 May 1984
TI Large scale isolation, purification, and partial characterization of the intermediate filament-specific, calcium-activated proteinase from porcine kidney and Ehrlich ascites tumor cells: a comparative study
AU Traub, Peter
CS Max-Planck-Inst. Zellbiol., Ladenburg/Heidelberg, D-6802, Fed. Rep. Ger.
SO Archives of Biochemistry and Biophysics (1984), 228(1), 120-32
CODEN: ABBIA4; ISSN: 0003-9861
DT Journal
LA English
CC 7-2 (Enzymes)
AB The Ca2+-activated, neutral thiol proteinase specific for intermediate filament subunit proteins was isolated on a large scale from the postribosomal supernatant of a low-ionic-strength extract of porcine kidney and Ehrlich ascites tumor (EAT) cells. The purification procedure encompassed DEAE-Sephacel ion exchange chromatog. of the material precipitating between 23 and 55% (NH4)2SO4 saturation, followed by hydroxylapatite chromatog. and activated thiol-Sepharose 4B covalent chromatog. On the average, 25 mg of 62% pure enzyme was obtained from 500 g frozen kidney and 55 mg of 51% pure enzyme from 500 g EAT cells within a week. Both enzyme prepns. were free of Ca2+-independent proteolytic activities and indistinguishable with respect to their physicochem. and functional properties; their catalytic properties were indistinguishable from those of enzyme purified to homogeneity on arginine Me ester-Sepharose 4B. Because of this identity, porcine kidney proves to be an inexpensive source for the Ca2+-activated proteinase which had previously been isolated and purified on a small scale from EAT cells (Nelson, W. J.; Traub, P., 1983). Despite a 38% protein contamination, the partially purified enzyme from porcine kidney is useful for the in vitro study of structure-function relationships of intermediate filaments and their subunit proteins. During affinity chromatog. of the partially purified proteinase from EAT cells on arginine Me ester-Sepharose 4B, a 100-kilodalton protein was purified which has a high affinity for arginine residues. It also occurs in porcine kidney, although at a considerably lower concentration Its cellular localization and function remain to be determined
ST intermediate filament proteinase kidney carcinoma; Ehrlich ascites intermediate filament proteinase
IT Kidney, composition
(intermediate filament-specific proteinase of)
IT Proteins
RL: BIOL (Biological study)
(of intermediate filaments, proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)
IT Vimentins
RL: BIOL (Biological study)
(proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)
IT Carcinoma
(Ehrlich ascites, intermediate filament-specific proteinase of)
IT Proteins
RL: BIOL (Biological study)
(arginine-binding, of Ehrlich ascites cells, intermediate filament-specific proteinase in relation to)
IT Microfilament and Microtubule
(intermediate filament, proteins of, proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)
IT 78990-62-2
RL: BIOL (Biological study)
(of Ehrlich ascites cells and kidney, large-scale isolation and purification

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(arginine-binding, of Ehrlich ascites cells, intermediate filament-specific proteinase in relation to)
IT Microfilament and Microtubule
(intermediate filament, proteins of, proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)
IT 78990-62-2
RL: BIOL (Biological study)
(of Ehrlich ascites cells and kidney, large-scale isolation and purification

and properties of)

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and properties of)

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and properties of)

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